IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

OFFICIAL

Appellants:	Elizabeth M. Denholm, Elizabeth Cauchon, and Paul J. Silver		
Serial No.:	09/727,873	Group Art Unit:	1651
Filed:	December 1, 2000	Examiner:	M. Meller

For: *ATTENUATION OF FIBROBLAST PROLIFERATION*

Assistant Commissioner for Patents
Washington, D.C. 20231

REPLY BRIEF

Sir:

The following remarks are in response to the examiner's answer mailed February 23, 2004, in response to the Appeal Brief mailed November 28, 2002.

There appears to be an error in the Patent Office record - the appeal brief was not mailed August 8, 2003, but November 28, 2002. The file was reconstructed August 8, 2003, after numerous requests by the undersigned. Accordingly, the appeal has been pending since August 28, 2002, when the Notice of Appeal was originally filed.

21 (7) **Grouping of Claims**

The legal standard for whether the claims stand or fall together is not whether or not "the art either applies to all the claims or not".

The standard for whether the claims stand or fall together is whether or not there are patentably distinct limitations in the claims that distinguish over the art. There is no question in this case that the claims should not stand or fall together.

(8) **Arguments**

(b) **The claimed subject matter is not obvious under 35 U.S.C. 103**

Claim 1 is drawn to a method to decrease fibrous tissue size by administering to an individual in need of treatment an effective amount of a dermatan sulfate or chondroitin sulfate degrading enzyme to decrease fibrous cell proliferative response to growth factors, reduce secretion of collagen by fibroblasts, and thereby decrease the size of fibrous tissue.

The examiner has rejected the claims based on U.S. Patent No. 6,153,187 to Yacoby-Zeevi, which describes treating patients with a chondroitinase to treat cystic fibrosis. The examiner's rejection is based on a lack of understanding of the pathology of cystic fibrosis, presumably due to the name "cystic fibrosis".

The claims are drawn to a method of decreasing the size of fibrous tissue by decreasing fibrous cell proliferative response to growth factors and reduction of secretion of collagen.

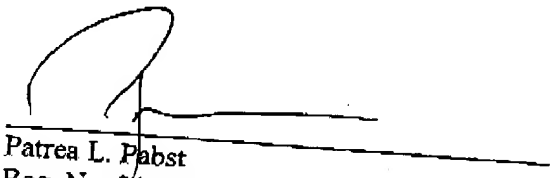
As demonstrated by the enclosed excerpts from the National Institutes of Health website, cystic fibrosis is not a disease of fibrous tissue. To the extent that chondroitinase is useful in treatment of cystic fibrosis, it is by virtue of decreasing the viscosity of the secretions that are abnormally thick in cystic fibrosis patients. This is consistent with the explanation in Yacoby-Zeevi at col. 6, lines 55-65, for example.

There is simply no teaching of administering a chondroitinase or any other glycosaminoglycanase to decrease the size of fibrous tissue.

Triscott just does not make up for this deficiency. Triscott is not a method of treating a patient, Triscott relates to a laboratory assay.

In summary, the prior art does not make obvious the use of glycosaminoglycan degrading enzymes to decrease fibrous tissue as claimed in claims 1-11.

Respectfully submitted,



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Cystic fibrosis

By Mayo Clinic staff

Overview

Cystic fibrosis (CF) — a life-threatening disorder that causes severe lung damage and nutritional deficiencies — used to be a genetic mystery, and most people with the disease didn't live beyond their teens. But researchers have made progress in unraveling the genetic basis of CF, which has led to earlier detection. In addition, improved and more consistent treatments now allow people with CF to live into their 30s and 40s and even beyond, and to have fuller, more comfortable lives.

CF is an inherited (genetic) condition affecting the cells that produce mucus, sweat, saliva and digestive juices. Normally, these secretions are thin and slippery, but in CF, a defective gene causes the secretions to become thick and sticky. Instead of acting as a lubricant, the secretions plug up tubes, ducts and passageways, especially in the pancreas and lungs. Respiratory failure is the most dangerous consequence of CF.

Each year approximately 3,200 white babies are born in the United States with CF. The disease is much less common among black and Asian-American children. Most babies born with CF are diagnosed by age 3, although mild forms of the disease may not be detected until the third, fourth or fifth decade of life. In all, about 30,000 American adults and children are living with the disorder. Although there's still no cure, the emerging field of gene therapy may someday help correct lung problems in people with CF.

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Cystic fibrosis

Signs and symptoms

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- Screening and diagnosis
- Complications
- Treatment
- Self-care
- Coping skills

The specific signs and symptoms of CF can vary, depending on the severity of the disease. For example, one child with CF may have respiratory problems but not digestive problems, while another child may have both. In addition, the signs and symptoms of CF may vary with age.

In some newborns the first sign may be a blockage of the intestines (meconium ileus). This occurs when meconium — tarry, greenish-black stools normally passed by an infant in the first day or two after birth — becomes so thick it can't move through the intestines. Other signs in newborns may include a failure to grow, bulky and greasy stools (steatorrhea), and frequent respiratory infections.

The signs and symptoms of CF in children and young adults may include:

- Salty taste to the skin. People with CF tend to have two to five times the normal amount of salt (sodium chloride) in their sweat. This may be one of the first signs parents notice because they taste the salt when they kiss their child.
- Blockage in the bowel.
- Foul-smelling, greasy stools.
- Delayed growth.
- Thick sputum. It's easy for parents to overlook this symptom because infants and young children tend to swallow their sputum rather than cough it up.
- Coughing or wheezing.
- Frequent chest and sinus infections with recurring pneumonia or bronchitis.
- Growths (polyps) in the nasal passages.
- Cirrhosis of the liver due to inflammation or obstruction of the bile ducts.
- Displacement of one part of the intestine into another part of the intestine (intussusception) in children older than age 4.
- Protrusion of part of the rectum through the anus (rectal prolapse). This is often caused by stools that are difficult to pass or by frequent coughing.
- Enlargement or rounding (clubbing) of the fingertips and toes. Although clubbing eventually occurs in most people with CF, it also occurs in some people born with heart disease and other types of lung problems.

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Cystic fibrosis

Causes

In CF, a defective gene alters a protein that regulates the normal movement of salt (sodium chloride) in and out of cells. This results in thick, sticky secretions in the respiratory and digestive tracts, as well as in the reproductive system. It also causes increased salt in sweat on the skin.

The affected gene, which is inherited from a child's parents, is known as a recessive gene. This means children need to inherit two copies of the gene, one from each parent, in order to have the disease. If children inherit only one copy, they won't develop CF, but they will be carriers and possibly pass the gene to their own children.

If two people who carry the defective gene conceive a child, there's a 25 percent chance the child will have CF, a 50 percent chance the child will be a carrier of the CF gene, and a 25 percent chance the child will neither have the disease nor be a carrier.

People who carry the CF gene are healthy and have no symptoms. In fact, it's estimated that as many as 10 million people may be carriers of a CF gene and not know it. Although parents often blame themselves when a child is born with CF, it's important to remember that nothing a parent does causes this disease.

In addition, some experts believe that an imbalance of essential fatty acids may play a role in cystic fibrosis. People with CF appear to have excessively high levels of arachidonic acid and a deficiency of another fatty acid, docosahexaenoic acid. Healthy people who carry one CF gene have fatty acid levels midway between those of people with CF and people with no genetic mutations for the disease. But the exact nature of the relationship between fatty acid levels and the gene defect that causes CF isn't clear.

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